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ON TUMORS INVOLVING THE CORPUS CALLOSUM.*

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The interest attaching to the subject of tumors involving the corpus callosum is not to be wholly measured either by the therapeutic advantage which a correct diagnosis can give us, which is very slight, or by the purely scientific problems that are involved, but also by the fact that there is considerable likelihood of mistaking the effects of such growth for functional psychoses of various sorts, and still more for the symptoms of general paresis.

Strictly speaking, neither of the three cases to be described—and the same can be said of those reported by others—are instances of pure corpus callosum tumor, since, as a rule, one or both hemispheres or the central ganglia were considerably involved at one or another point. Perhaps the only symptoms attributable to the injury to the corpus callosum itself are the mental changes, which will be discussed later. In spite of this tendency to the involvement of adjoining portions of the brain, however, these growths show, in certain parts, a distinct predilection for the corpus callosum. This is especially true for the posterior portions of the diseased area in our cases II and III, where the pigmentary discoloration of the tumor sharply defines the outlines of the great commissure.

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*Read before the American Neurological Association, June, 1901.

Case I. The first case was that of a patient who was under the care of Dr. Williams during a portion of her illness, and has been reported by him in the Boston Med. and Surg. Jour. for May 16, 1901.

The facts of chief interest were as follows: This patient was a woman, twenty-two years old. Active symptoms lasted about fifteen months, the first complaints being of headaches and vomiting, followed by vertigo and well-marked double optic neuritis. When examined one year later these symptoms had subsided and only obscure anemic signs were found, but the optic neuritis had been followed by a well-marked atrophy. After a number of months of quiescence, active symptoms recommenced, so that she was admitted to the Boston City Hospital. The hospital records read: "The pupils are equal and regular and respond slightly to light. Muscular movements are apparently normal. The patient states that she sees nothing with the left eye, and with the right only objects held to the extreme temporal side. Heart and kidneys are normal. There is no tenderness of extremities, no loss of power in arms and legs and no impairment of sensation." The optic nerve atrophy progressed so that vision was lost in both eyes before she left the hospital. Two weeks later she was admitted to the Carney Hospital in a serious condition. The records say that the left pupil was much dilated and neither responded to light. No perception of light was present in either eye. The right knee-jerk was absent. Sensation was apparently diminished over both legs and body up to the waist line. The muscular sense was impaired, and the sensation seemed to be "delayed in both arms;" but there was much mental dulness which probably accounted for the sensory disturbance. On the following day the pupils were somewhat contracted, but were equal and responded well to light. She died a few days later, but, meantime, optic neuritis was noted in each atrophied disc. Exitus in coma.

The points of special interest in this case were the temporary cessation of symptoms, and the fact that a well-marked optic neuritis developed at a time when optic atrophy had been for some time complete; and in this respect the case is almost unique. The autopsy, as performed by Dr. Magrath, showed a large tumor lying beneath the corpus callosum and reaching with its apex the optic commisure at the base of the brain. [Figs. I and II.] Serial sections of the hardened brain show that the tumor first appears at about 4 cm. from the anterior extremity of the right frontal lobe, being situated in the very front of the anterior horn of the right lateral ventricle. Here it is pear-shaped, the small end going over the median line to the left side, while the large end encroaches on the right hemisphere to within 3 cm. of its external surface. In so doing it occupies the whole of the right lateral ventricle, which has begun at this point. The greatest dimensions of the tumor were: width 6 cm.; depth 5 cm. The third serial section shows the head of the nucleus caudatus present. The cross-section of the tumor is now triangular-shaped, one corner reaching to the base just in front of the optic commisure. At this level the corpus callosum appears as a thin membrane above

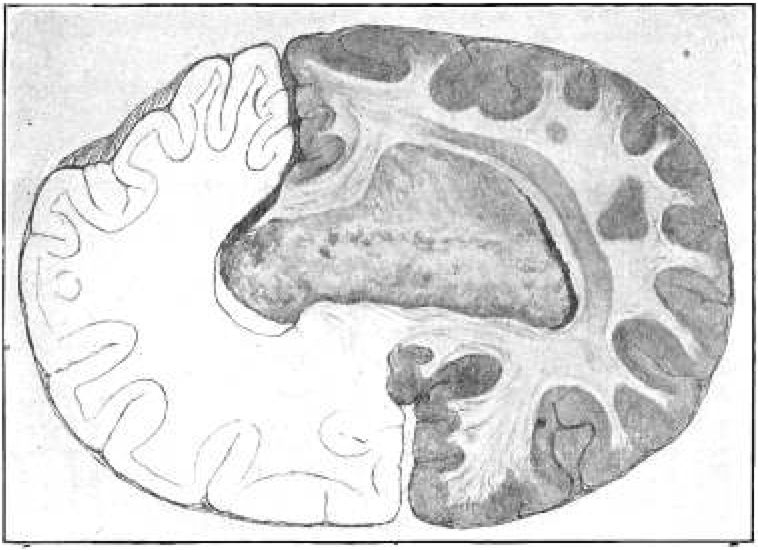

Fig. I. The corpus callosum was here cut through at the autopsy on the left side.

the tumor. Each side of the triangle is about 5 cm. long. The median side is shorter than the other two, so that the external corner blocks the right lateral ventricle.

The fifth section [Fig. II] shows the tumor now fairly extending to the base and completely compressing the optic tract. With its apex it actually projects into the interpeduncular space, and in so doing compresses both third nerves, especially the left one.

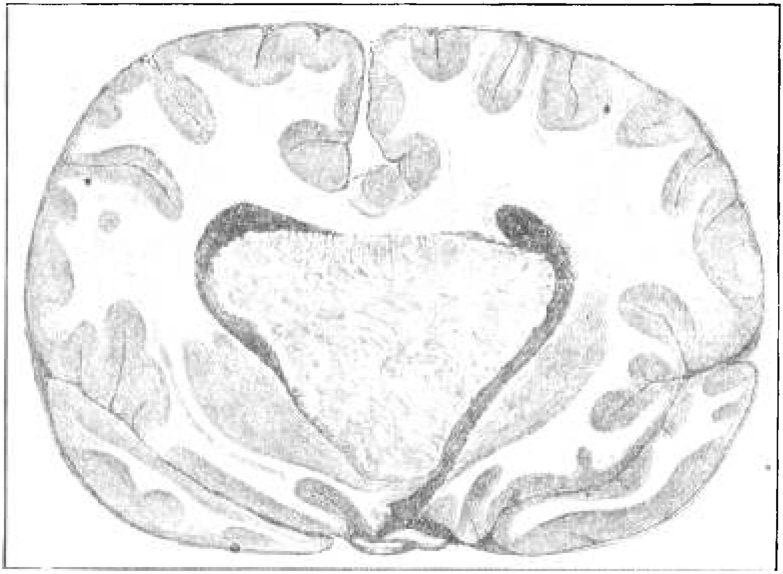

Fig. II.

The sixth section nearly bisects the pons. The tumor appears now only as a grayish mass which here occupies the floor of the right lateral ventricle and is not seen at all in this section on the left side.

Case II. This case was that of a gentleman of fifty, with a good record as regards health and mental vigor, except that he had been somewhat liable to "faint turns," when slightly ill from any cause. Three or four years ago he had fallen to the ground in such an attack, which was brought on perhaps by diarrhea. It may be that these were in fact epileptic attacks, such as have occurred in the early history of other cases of this same sort. From this point of view they are of marked interest as pointing to the possibility of a very early beginning of the new growth. There was no history of any severe illnesses, or injuries, or of any exposures which would have been likely to cause the final disorder.

He consulted Dr. Putnam on the 5th of August, 1898, and then stated that he had considered himself well up to the middle of June of the same year, when he was taken down with what he called "nervous prostration." He had done business up to June 29th, but his wife stated in response to close inquiry that he had been "rather nervous" for two or three years, and more irritable than formerly. These symptoms had been worse for some months, and had been associated with a vague sense of pressure in the head and eyes, and an unusual inclination to lie down and rest. On one occasion he had had a "numbness" of the right leg, which lasted a day or two and then passed away. This had come on suddenly, but was not attended with loss of consciousness. It also appeared that his mental characteristics had gradually undergone a certain amount of change, of a kind which suggested the possible onset of general paresis. Thus, in June, he had insisted, as a mere whim and against the advice of his carpenter, on having an elaborate bicycle-house put up behind his own residence. His plan of construction for this was poor, the position selected was unsuitable, and he was finally induced to abandon the enterprise in part. His business partner had noticed a falling off in his judgment, and a tendency to hastiness in small matters. Thus, on one occasion, he had rushed off and bought a lot of wheat, without sufficient reason, and on another he had ordered a quantity of coal for his sister, without having been asked to do so, on the plea that it could be bought a little cheaper than usual. For the past six months he had felt no inclination for recreation of any sort. Letter writing had become difficult, and he frequently repeated himself both in speech and on paper, and occasionally left out words. Throughout the spring he had been troubled about sleeping, and, partly on this account, partly because he found it more and more difficult to work, he had been persuaded to go on a trip to the Rangeley Lakes. There he at first improved, but after a few weeks his right hand became paretic and at the same time his speech grew somewhat difficult, although, as on the occasion when the leg became numb, there was no apoplectic seizure. His ability to collect his thoughts had continued to fail, and his mind worked slowly. Both legs had become somewhat weak, but it was the right hand that mainly gave him trouble.

On physical examination he was found to be an intelligent-looking man, though but little inclined to take part in the conversation which went on around him, either with word or expression of the face. His speech was hesitating and stammering in a high degree. He was unable to give the name of the town and country where he had stayed for a long time during the previous month, and made various mistakes in telling the story of his illness. The right hand and right side of the face were paretic; the tongue and lips trembled. The knee-jerks were normal, and the pupils were equal and reacted well to light. The tongue was coated; the bodily temperature normal; the heart's action normal. In spite of the awkwardness of the right hand, he was able to write clearly and fairly well, but omitted letters here and there, a sign which seemed to bear out the notion of a diffuse degeneration of the brain-cortex. Ophthalmoscopic examination failed, at that time, to reveal any sign of optic neuritis.

The patient was not seen again until about two months later, and by that time a marked change had taken place in his condition. He was now suffering from unmistakable signs of brain tumor—headache, nausea, vomiting, optic neuritis—and besides this the speech had become slower and more hesitating, and the difficulty in the use of the right hand had increased. There was also an awkwardness of both legs, so that he toppled over twice in going up stairs. The memory was considerably impaired. The knee-jerks were now exaggerated.

With a view to at least mitigating the effects of pressure, the patient was advised to enter the Massachusetts General Hospital for operation. Here he came under the care of Dr. J. C. Warren, who on November 8th, made a large trephine opening on the left side over the Rolandic area, but without opening the dura, which was, however, found to be very tense. The operation was done under chloroform[1], but in spite of this precaution the shock was so great that for several hours it seemed probable that the patient would die. After the effects of this shock had passed away slight improvement began to show itself, both as regards the general symptoms, the speech, and the use of the limbs.

This improvement did not go very far, however, and after lingering for two months, practically unable to speak or to help himself though not actually paralyzed, and in a condition of considerable mental dulness or apathy but without pain or coma, the patient died.

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[1] As specially advised by Horsley for cases of operation for increased intracranial pressure. In a case of this sort observed by Dr. Warren and myself the patient, a young girl, died after the use of a very moderate amount of ether and before any incision had been made. Artificial respiration kept the heart beating for five hours, but in vain.

The autopsy was made by Dr. Magrath, and the brain placed in a formalin solution for preservation. After hardening a number of frontal sections were made, at intervals of 2 cm., with the following result: The tumor appears first at about 4 cm. from the anterior extremity of the left frontal lobe as a blackish mass which lies towards the mesial surface near the anterior extremity of the anterior horn. In the next section, about 2 cm. further back, it is of triangular shape, each side being about 6 cm. in length. It lies mainly in the frontal lobe, above the corpus callosum, but invading it in that portion of its course which lies in the left hemisphere from the mesial line outward. [Fig. III.] It also stretches somewhat across the mesial line into the right frontal lobe. The fourth frontal section cuts across the optic commissure. Here the tumor has a bilobular shape, the larger lobe occupying about one-third of the cut surface of the left hemisphere. The right lobe is only about half as large and extends across the corpus callosum well into the right hemisphere. The left lateral ventricle is entirely obliterated at this point and the right ventricle is almost obliterated. The basal ganglia, on both

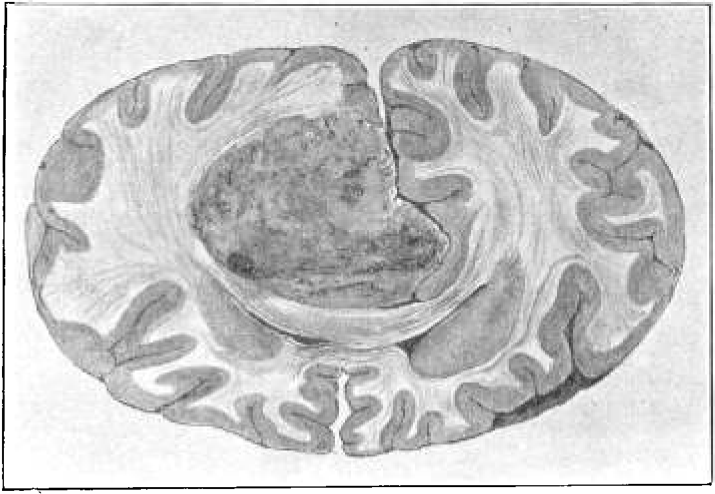

Fig. III.

sides, are very much compressed. The tumor extends to within about 0.6 cm. of the base, where its point comes opposite to the optic commissure. On the 6th section [Fig. IV], which transects the posterior portion of the thalamus, the tumor seems to be confined to the corpus callosum, but with a few outlying portions in adjacent parts of the left hemisphere also. The internal capsule is compressed but not invaded. The last section, which cuts across the posterior horn of the left lateral ventricle, reveals the last traces of the tumor, which is thus seen to involve the corpus callosum in its whole length, and to be almost confined to that body in its posterior half.

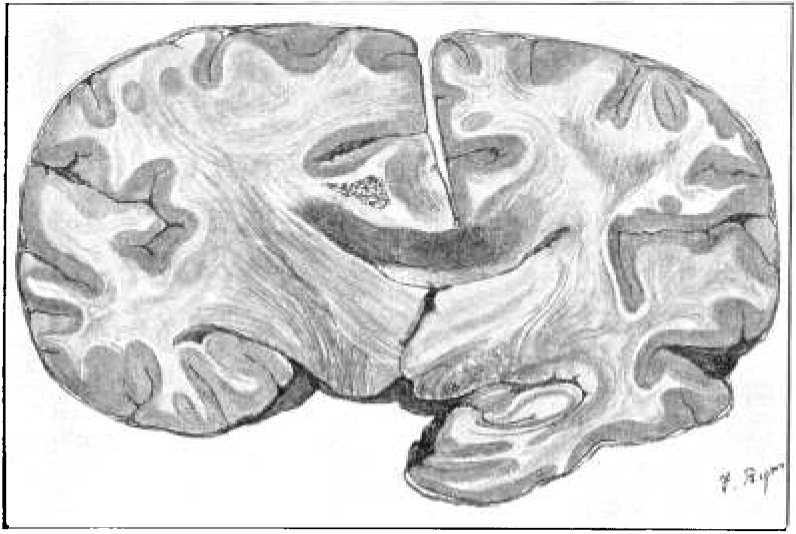

Fig. IV.

Sections for microscopical examination were made under the skilful supervision of Dr. E. W. Taylor, to study the question of secondary degeneration in the neighborhood of the tumor and in corresponding areas of the cortex. The method of hardening had been such that the use of the Marchi stain was out of the question, but no gross lesions such as could be shown by Weigert hematoxylin stains were discovered at any point. This was true also of the previous case, No. I.

The tumor itself was of gliomatous character in both cases.

The point of special interest in this case is the early appearance of slight changes in character and slight impairment of mental power and of memory, coming at a time when neither signs of focal lesions nor of general pressure had shown themselves.

The very early occurrence of isolated epileptic seizures several years before is also noteworthy, because, although it is not easy to explain them, the histories of several other cases[2] report similar incidents.

The paralysis, when it did appear, must evidently have been due to pressure, for which there was abundant cause in the presence of the mass on the left side.

Schupfer, whose observations will be discussed later, thinks that when the tumor is situated anteriorly as regards the corpus callosum the arrangements for the face and arms are more likely to be damaged most; when more posteriorly, those for the legs. The present case would perhaps bear out that view, but it is to be remembered that the relatively fine movements of the hands and of the muscles of speech are more apt to suffer from a given pressure than the movements of the legs. The weakness of the legs in this case, when it did occur, was relatively diffuse, and bilateral in distribution, as in the following case.

Case III was that of a patient under the care of Dr. Samuel Breck[3] with whom he was seen in consultation by Dr. Putnam a few days after the onset of his illness. He was a man 56 years old, strongly addicted to liquor, but of vigorous health. The first indication that anything was wrong was noticed on November 18th, 1899, when his conduct was seen to be peculiar and he was observed to be very slow in carving at the table. From this time the signs multiplied quickly, and he died on December 8, only about three weeks after the appearance of the first symptoms. On the first examination he was found to show a weakness of movement of the left leg. This soon spread to the left arm, and then to some extent to the right leg, but did not involve the right arm at all, or but little. For several days after the weakness of the legs began he was still able to walk and to empty the bladder voluntarily, but after the fourth day he was confined to bed and began to suffer with incontinence of urine and feces.

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[2] See table.

[3] We owe Dr. Breck our thanks for his kind permission to publish

The mental disturbances, which had begun with dulness and odd behavior, became more and more marked, though his downward course in this respect was broken by relatively clear intervals.

At times he would seem delirious, and would talk about his wife, who had been dead for many years, as if she was present. For the last ten days he was unable to feed himself, but would chew and swallow anything put into his mouth, and would sometimes indicate that he knew what he was eating. Otherwise, he never spoke.

It is important to note that the knee-jerks began early to diminish and finally disappeared, and also that during the few days, when he was still able to walk about, the gait was uncertain and unsteady.

The autopsy was made by Dr. Magrath, and the report here quoted on the position of the tumor was written by Dr. E. W. Taylor, after the specimen had been hardened in 10% formalin solution.

"Frontal sections approximately 1 cm. thick were made throughout the brain with the following result: The tumor first makes its appearance at a distance of about 2½ cm. of the right hemisphere, invading the brain substance and filling the longitudinal fissure at about the level of the corpus callosum and anterior to it. From this point the tumor increases in size toward the posterior part of the brain. The right hemisphere is invaded by the growth, the left not, although somewhat indented by pressure. At about the level of the top of the temporal lobes the tumor has attained its maximum size. Its shape is that of a wedge, its base lying in and involving the corpus callosum, its apex extending to within 1½ cm. of the upper surface of the brain in the longitudinal fissure. The caudate nucleus is not invaded. At a level immediately anterior to the optic chiasm the corpus callosum is almost completely destroyed by the new growth, the corpus striatum is not involved, and the mesial surface of the right hemisphere only to a slight degree. From this point the tumor diminishes rapidly in size, involving the corpus callosum completely and infringing only in the slightest degree on the right hemisphere as before described. The fornix is involved for a part of its course, as it lies opposed to the corpus callosum, otherwise no structures of importance are invaded. The tumor does not extend behind a section through the anterior portion of the thalamus. Neither the lateral nor third ventricles are involved in the growth. The capsules are intact, and there is no very marked evidence of pressure. The greatest size of the growth is about 4 x 4 cm. It is infiltrating, though its extent may easily be made out. There are various discolored areas in the mass of the tumor-necroses."

The report made at the time of the autopsy says that the left half of the tumor was soft and in places hemorrhagic, but not broken down; the right (larger) half had broken down into a gelatinous mass containing much old blood pigment.

The microscopic examination of the fresh specimen showed many round cells, mostly fatty, free fat, old blood-pigment and granular debris.

This case affords a striking example of latency, with eventually rapid outburst of symptoms. Even here, however, the damage to the brain-mechanism revealed itself first through disturbed mental action. In this case, too, both legs and one arm are said to have become weak or awkward without any great involvement of the other arm. Presumably, as in case II, the involvement of the legs came from pressure on the paracentral lobule. This extreme latency of symptoms has been noted in several cases of this class, as occasionally, of course, with tumors in other localities, perhaps especially the frontal lobes. In Zingerle's case, the time during which symptoms were present covered only seven weeks. Obviously, it is the rapid growth of the tumor or increase in its size through hemorrhage, or edema of the brain due to the involvement of vessels, that underlies this rapid development of symptoms. But, as regards the mechanism of the collapse itself, it is our belief that it is due in part to psychical causes. In other words, the strong instinct to reorganize and make use of compensation in every conceivably available form, and to maintain the same sort of front as before, even with a failing number of available cerebral reactions, is due to the activity of memory and constructive imagination, which are in a sense independent of the physical life of the brain and are constantly pressing for expression on old lines. When the time comes, however, that this expression is no longer even tolerably possible, the attempt at reorganization is abandoned and collapse rapidly follows.

The tumor itself in this case was almost the counterpart of that in the preceding case (II) but smaller, and involving the right hemisphere more than the left. In both cases the growths reached forward somewhat beyond the anterior extremity of the corpus callosum and invaded the structures of one or the other frontal lobes, and the question arises whether it may not have been to this fact that the mental disturbances were due.

The study of this group of cases falls naturally into two parts, the one purely clinical or diagnostic, the other physiological and psychological.

The diagnosis of tumors of the corpus callosum has been fruitfully studied by a number of writers, chief among whom are Bristowe, Giese, Ransom, Bruns, Zingerle, Schupfer.

It is unnecessary, in view of the thorough compilations made by these writers, to give other references in this paper, our object being only to indicate the movement of thought and discovery. The aim of the clinical diagnosis, as noted at the beginning of this paper, is not to distinguish tumors confined to the corpus callosum, but rather tumors which develop with that structure as their central point.

The conclusions reached by these various writers agree in essential respects. The usual pressure-signs of tumor are long delayed, and of positive symptoms, the mental disorder is the one of greatest interest. As stated by Ransom, this takes the form of a progressive failure; or of rapidly developing stupor; or of hallucinations, irritability or attacks of excitement if the progress of the case is not too rapid.

The paralysis, when it is present, is usually primarily hemiplegic and of the ordinary type, but becomes eventually more or less diplegic, at least as regards the legs, and this is highly characteristic, though doubtless due to pressure on conducting tracts lying outside the corpus callosum. Convulsions may occur and these too are liable to be bilateral.

On the other hand, the usual "general" or "pressure" symptoms of brain tumor often fail to appear until relatively late, and cranial-nerve paralyses are usually wanting. The tendon reflexes[4] may become exaggerated, but may be but little changed.

Zingerle calls attention to the frequency of a lack of power of balance, sometimes amounting to considerable incoördination, to which he gives the term "callosal ataxia;"[5] and to a lack of motor-initiative, or "poverty of movement" (*Bewegungsarmuth*), both of which symptoms have been described by early writers, though in other terms and with less insistence.

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[4] See Case III of this series.

His account recalls the helpless, apathetic way in which patient No. II of this series lay for the last two months of his life, not in pain and not incapable of considerable movement, nor, apparently, of more or less intelligent observation. but almost without a spontaneous sign or motion, except that he would follow about more or less with the eyes.

Schupfer attempts a subdivision of the corpus callosum tumors, and says that a growth at the level of the "*knee*" may be suspected; (1) when the disorders of motion that appear have been preceded for a considerable period by mental disorders; (2) when the lower facial muscles of one or both sides become paretic, independently or relatively early; (3) when the head turns toward the paralyzed (hemiplegic) side, or, in general, when there is contracture of the neck and head muscles; (4) when the paralysis of the arms is relatively greater than that of the legs; (5) when disorders of gait are present which recall cerebellar ataxia, though when these signs are associated with vomiting and vertigo they are more apt to be due to tumors lying posteriorly, in such a position that they compress or damage the cerebellum.

When the middle portion of the corpus callosum is the seat of the growth, vomiting is, he thinks, often absent or only of occasional occurrence, and the weakness of arms and legs is contemporaneous and at first of slight degree. Tumors lying posteriorly, in the *splenium*, are more apt to cause impairment of motion of the legs before the arms, the face remaining unaffected. In such cases uncertain and reeling gait, such as has been called frontal ataxia, occurs late. In general the symptoms in these cases suggest cerebellar disease, but the optic neuritis is of less rapid development. The mental symptoms occur either contemporaneously with the paralyses or later. Visual disorders (occipital) would be likely to occur, but have not been noted, and might sometimes be masked by those dependent on the neuritis. So, too, the mental disturbances would, he believes, often mask an ataxia which Redlich thinks might be expected as a result of tumors of the splenium.

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[5] This is the sign referred to by Zingerle, and called by Bruns "frontal lobe" ataxia. Giese considers that these ataxias are really of cerebellar origin.

In order to base our clinical conclusions on the greatest available number of facts, we have prepared a summary of all admissible cases hitherto published, amounting to 38[6].

An analysis of this table justifies the following statements as fairly though not absolutely reliable.

The whole number of cases in the table is thirty-eight, but in about twelve of these the growth was mainly, though by no means absolutely, confined to the corpus callosum, and these twelve have been analysed apart from the rest for the purpose of comparison.

Both groups show the marked prevalence of mental changes and, in general, their early occurrence. Thus, of the thirty-eight cases, mental changes of one or another kind occurred very early in thirty-four at least; while of the smaller group of twelve selected cases some sort of change of character or mental disorder was present in all but two. Schupfer (*l.c.*) gives an interesting discussion of this tendency, and makes out that of the twenty-five cases collected by him some sort of mental change was universally present, sooner or later. In contrasting these cases with those of tumors in other parts of the brain, he cites the authority of Giannelli, who found mental disorders, with tumors of the frontal lobes in 81% of a large number of reported cases; with tumors of the parietal lobes in 50%; of the temporo-sphenoidal lobes in 66%; of the occipital lobes in 60%; with diffuse neoplasms in 59%; with tumors of the basal ganglia in 50%. The significance of these facts, as bearing on the general subject of the cerebral functions, will be discussed later.

Alongside of the mental changes, the "physical inertia," or lack of physical initiative, deserves mention, since, although referred by Zingerle to diminution in number or force of centripetal impulses, it might be due as well to mental lethargy or anesthesia.

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[6] Erb reports a case of hemorrhage into the corpus callosum, which is reported in our list but not included in the summary and analysis.

At all events, it is a symptom on which future inquirers must pass judgment, since it was only positively referred to as present in nine out of the thirty-eight cases, and in one of the selected twelve cases. Paralysis of the limbs, of hemiplegic distribution, though not always involving arm and leg with equal severity, was present in fourteen of the thirty-eight cases, and in four of the selected cases. Bilateral paralysis, which is the far more significant sign, was present in nine out of the thirty-eight cases, but in only one of the twelve selected cases where the tumor was mainly confined to the corpus callosum. This is not remarkable, since the paralysis, whether unilateral or bilateral, is doubtless an affair of involvement of the hemispheres. In two cases the face was paralyzed alone, and in one the tongue was reported immovable. In another case the face was not paralyzed, but was the seat of twitchings on the left side, and the head was drawn round to the right. In all these cases the tumor lay anteriorly, and the localizing rule given by Schupfer is thus in a measure confirmed, although, as might be expected, the tumors lying posteriorly may cause a paralysis of the face together with the limbs. The term "paralysis" does not adequately indicate the motor disorders observable in these cases. "Weakness" and helplessness (compare remarks on incoördination) were common signs, and have been noticeable as regards the legs. So too "rigidity" or contracture, either with or without obvious paralysis, is occasionally noted, and in one case, where no paralysis, strictly speaking, was observable, the patient could not raise the legs from the bed while in a recumbent position.

As regards the *character* of the early mental changes, it is noteworthy, to begin with, that no positive line is to be drawn between the cases where the tumor lay relatively anteriorly and those where it lay rather in the median or posterior portion of the great commissure, though, in fact, the anterior portion seems to have been involved more frequently than the posterior in those cases where the tumor was mainly confined to the callosal tracts. As a rule, impairment of memory, dulness, and intellectual failure were the marked features, but many times changes are reported which strongly suggest paresis—as foolish or irrational behavior, irritability and indifference.

Where alterations of the emotional tendencies were present these were far oftener of excitative than of depressive character, and outbreaks of violence and even maniacal excitement now and then occurred, perhaps supervening on more chronic forms of degeneration. Ransom, writing in 1895, says: "In all the slowly-developed cases excitability was marked, maniacal outbreaks being common." In the summary he speaks of "Gradual but marked mental change, in the more acute cases taking the form of increasing stupor, in the more chronic appearing as insanity, with delusions, irritability and maniacal outbursts."

There is no explanation, at present, for this predominance of excitability over depression. At all events, the relative frequency of these emotional states is perhaps much the same for these tumor cases as for cases of "general paresis," which they so often simulate. Perhaps the most striking fact of all is that mental changes of all kinds may remain so long absent, or may even not occur except as practically premortal symptoms. Examples of such cases are those reported by Gläser, and those like our third case and Miss Blackwell's and various others, where the final symptoms even though largely mental were of but brief duration. Here, too, belongs a reference to the oft-cited cases of congenital absence of the corpus callosum unattended by distinct symptoms, and to the experiments upon dogs, showing that at the least motor paralysis need not result from longitudinal section of the commissure.

These facts are to be stored up, not as proving that the corpus callosum fulfils no useful function, but as indications of the rich resources of the brain in the way of compensation. The experiments of R. Ewald are here to be borne in mind, which indicate that the remarkably rapid, and sometimes almost instantaneous recovery from the effects of localized cortical ablations implies the integrity of the coördinating apparatus of the auditory labyrinth, and fails to occur if the latter has been damaged.

It is also to be remembered that in the daily routine of life few persons are tested according to the highest standards. Most individuals of the age at which these tumors usually cause symptoms are doing work of a sort far inferior to that which might have been expected of them if their brains had ever been trained to their best accomplishment, and generally inferior to that which they have done in the past. Their machinery is damaged, and the fact would be patent if they were put to a severe test. It is in these two facts, the immense fertility of resource of the brain in the line of compensation, and the relatively slight test of efficiency which is usually applied, coupled with the third fact, so often misappreciated, that memories are not "stored in the brain," but remain independent, and press for materialization and expression, if only *some* machinery for even passably adequate expression can be found, that the explanation for the latency of symptoms due to partial or even complete destruction of the corpus callosum is to be sought.

Equally interesting with the latency of symptoms is the fact that *apparently* isolated indications of the presence of these tumors may show themselves long before the case fully declares itself. Thus, in our case II and in the cases reported by Ransom, and by Devic and Paviot, isolated epileptic seizures had occurred several years before the final outbreak. Again, in Miss Blackwell's case, the patient's history previous to the final and brief illness, only five weeks in duration, had been marked by several short attacks of mental derangement, to which, it should of course be said, some hereditary tendency was present. It is needless to say that these facts are open to another explanation than that of the presence of the callosal disase, and that even if due to that cause the mode of production of the symptoms in question would probably be usually classified as either reflex or nutritional.

This latter remark applies likewise to the epilepsies, contractures, tremors and other irritative motor symptoms which occasionally occur in later stages, provided that opportunity for direct pressure is absent.

General tumor symptoms, that is, headache, nausea and vomiting, occurred early in 17 cases of the larger group, and late in 5 cases. In 5 cases these symptoms were altogether wanting. Of the smaller group of selected cases the general symptoms appeared early in seven. It is worth noting that the knee-jerk is occasionally diminished, although perhaps this tendency is not so marked as Ransom thought it to be. *Convulsions* without very definite characteristics were present in 16 cases out of the 38, and in 6 out of the selected 12.

Optic neuritis was noted as present in 15 cases and absent in 7 cases of the larger group; present in 4 cases and absent in 2 cases of the smaller group. In the rest of the cases the sign was not referred to.

The condition of the pupils is too variable to be of special interest.

Other *cranial nerve* complications are usually lacking, for obvious reasons, but the situation of the tumors in several cases (see cases I and II) is sometimes such, on the other hand, that the nerves of the base, and especially the optic chiasma are subjected to direct pressure.

The interesting fact, illustratd by case I, that even after atrophy due to this pressure, an optic neuritis may occur associated with other "general" signs of cerebral tumor, is of striking interest.

The clinical course of these cases is important mainly from its occasionally striking brevity, as is shown by the following table:

	Whole 38 cases	12 selected cases
Under 1 month.....	7	3
1 to 2 months.......	3	0
2 to 3 months.......	5	1
3 to 4 months......	2	2
About 1 year.......	5	1
1 to 2 years.........	1	2
2 to 5 years........	2	1
About 15 years......	1	0
Doubtful...........	12	2
	38	12

Finally, it would be germane to inquire whether the analysis of this larger body of cases bears out the newer diagnostic inferences of Zingerle and of Schupfer, the former of whom laid stress on the importance of a sort of ataxia of gait, suggesting the cerebellar reel, and of a marked physical inertia, the latter on the possibilities of differentiation of tumors situated relatively towards the anterior, the middle and the posterior portion of the corpus callosum, respectively, by the diagnostic points cited above.

Our attitude on these questions is that the cases that have been carefully studied with special reference to these points are as yet too few to justify an opinion, but that future examples should be thoroughly observed in these respects.

ANATOMY AND PHYSIOLOGY.

A good summary of what is known on the anatomy and physiology of the corpus callosum is given in Schäfer's excellent discussion on the cerebral cortex, in his Text-book of Physiology, published in 1900. It should be said, however, that the reliability of the important observations by Mott and the author himself which are cited in the work mentioned as also in every publication on this subject, has been rendered doubtful by the recent experiments of Lo Monaco, to which reference is not there made.

Mott and Schäfer had, namely, found that excitation by electricity, either of the upper surface of the corpus callosum or of the surface exposed by longitudinal section, induced definite movements of the trunk and limbs, etc., varying with the position of the electrodes. Lo Monaco, on the other hand, finds the corpus callosum inexcitable by electricity, though his experiments were done by what is claimed to have been a superior method.

Leaving this question undecided, it is at present undisputed that longitudinal section of the corpus callosum produces, in dogs, no paralysis, and no easily apparent change in the disposition or intelligence, and further, that although the corpus callosum is a true commissural tract, yet the neurone-processes which pass there do not connect strictly symmetrical areas of the two hemispheres, but scatter as they proceed.

This observation is significant, because if it is true it becomes clear that we cannot assume that the existence of the corpus callosum implies a simultaneous, or "twin" action of homologous areas of the brain-cortex of the two sides.

The very latest investigation with regard to this point, that is, with relation to the course of neurone-processes which connect the two sides of the brain, as indicated by degeneration-experiments, is recorded by E. Lindon Mellus of Johns Hopkins, in a paper published in the recent issue of the Johns Hopkins Bulletin. This observer, working by the Marchi method, finds that the degenerating fibers running from limited areas of the cortex, can be traced far more widely than has hitherto been demonstrated.

Reference can not here be made to the great number of highly interesting details given in this paper, which prove how purely relative is the significance of cortical localizations made through the use of electricity. Suffice it to say that as a result of the removal of pieces of cortex one centimeter in diameter from the "hand-center" of the Rolandic convolutions, signs of degeneration were found over a considerable portion of the corpus callosum. It is also noteworthy "that distribution of association fibers to the convolutions of the two hemispheres is very nearly equal and quite symmetrical."

Inspection of the writer's diagrams shows that the cortex over more than the whole "motor" area is involved. The distribution of the degeneration "extends also upon the internal (mesial) surface of both hemispheres as far as the calloso-marginal fissure."

It has been generally believed that the corpus callosum does not carry neurone-processes destined for the internal capsule and peduncle of the opposite hemisphere, though it is held by Ferrier and Turner as well as by Zingerle that it is a pathway for corticipetal fibers from the thalamus.

Schupfer, in his excellent paper, discusses several fine points of anatomy relating to the corpus callosum, and shows that the tapetum is not to be reckoned as a part of this commissure, but these questions we cannot now take up.

The tumor described by him invaded a portion of the thalamus, and it is to this fact, he thinks, that several of the most striking symptoms were due.

The fact that the corpus callosum is not necessary for the performance of the grosser functions of the brain-life is shown not only through experiments on animals, but also through the oft-cited cases where it was congenitally absent, and through those observed respectively by Erb and by Kaufmann, where no very definite symptoms attended its partial destruction through hemorrhage or softening.

No one would be tempted to assume, however, in spite of these observations, that this great commissure exists for nothing, and they stand as one challenge the more for us to find out what part it does subserve, and in what manner its absence is ordinarily made good.

To do this with positiveness may be impossible, but we can not even begin to understand such a question without adopting more clarified views than are now current as to the fundamental nature of the part played by the brain in the economy of the conscious life.

Although it may be true that the typical psychoses are at present best studied, as is often maintained, not from the standpoint of psychology but as clinical entities, that is not the case here where a typical course is not in question. So long as we cling to the vague, crude, and misleading idea that the brain is, in any proper sense, the repository of the mental functions, so that, for example, the cortex is rightly to be spoken of as a store-house of memories, we shall remain at sea in the analysis of mental disorders related to definite brain-injuries such as are to be dealt with in these cases.

Two comparatively recent books are of prime importance in this connection, the one by Loeb, on the physiology of the brain, the other by the psychologist Bergson, on the nature of memory in its relation to the physical world, so to speak, of which the brain is strictly a part. Loeb's observations make it strikingly clear that the functions of the nervous system, including the brain, can profitably be studied as representing only the marvelous working out of modes of reaction already inherent in all living protoplasm. Bergson's work, although not experimental, but speculative and analytic, goes, in its own path, far deeper, and shows, by a process of reasoning of extraordinary subtlety, that the brain would occupy no privileged position over anv other portion of the nervous system or indeed of the whole physical universe, in respect to the mind, were it not for the fundamental fact that it represents a mechanism capable of responding to a given stimulus, not always in the same way, but in any one of numerous different ways, and even in these different ways all at once, if the nascent responses, most of which come to nothing, are taken into account.

Memory, which is the primal and fundamental mental function, linking together the past and the future, and clothing perception with all that gives it color and value, is in a certain sense independent of the brain, though it would, of course, never exist but for the fact that the brain responds in the way it does, and cannot come practically and definitely to the service of the individual except through the the intervention of brain-activity.

The brain, then, is to be studied solely as a piece of machinery, and the greater part of the disorders of memory due to defects in its operation are broadly classifiable under one of two heads, according to whether the lesion is one that affects the general vitality of the brain as a whole (toxic and nutritive disorders), or is a local destruction of brain elements.

In the former case no special memory may be lost, but the relations of all of them to the outward life of the individual will be less firm, and a veritable "rupture of the mental equilibrium" (impairment of sense of identity, etc.) may result. In the latter case the mental equilibrium may not be broken, but the memory will lose something of its complexity. The individual may present the same *sort* of front towards the world, mentally speaking, as before, and will strive to effect a reorganization of his functions on the old lines, but he will be like a person trying to work with less good tools than before, or to do without some special tools; and from this strange results may ensue.

The recent work upon the functions of the frontal lobes as compard with the rest of the brain, is interesting in this connection. Thus, for example, Bianchi's reasoning, while it confirms the doctrine that mental disturbances pre-eminently attend frontal-lobe disorders, yet assigns to this portion of the brain no specific functions, but only that of gathering together again the same sensori-motor mechanisms which have already been utilized for simpler purposes, and combining them for new ends, at once more comprehensive and more specific.

One might, perhaps, use the simile that by the aid of the frontal-lobes we think and act relatively on the basis of symbols which condense many details into a brief expression, just as the mathematician condenses a mass of figures for more convenient use by the aid of the differential calculus. It is not however, to be assumed that it is in the frontal lobes alone that this kind of mental operation goes on, but only that without them it cannot readily go on. Again, the very recent investigations of Tonnini on the comparative effects on the mental functions of removing the cortex of different parts of the brain, show that this operation is followed by closely similar results, no matter whether done on the frontal lobes or on other portions, but that a relatively small removal of cortex from the frontal lobe is equivalent to a large removal elsewhere. It is not, therefore, necessary to assume that the mental disorders which attend the development of tumors of the corpus callosum imply always an interruption between the two frontal lobes, or a cutting off of one frontal lobe from its normal connections with the rest of the brain, though such lesions as these might be pre-eminently effective. Neither can one expect to explain, on anatomical grounds, why hallucinations and excitement are characteristic of one case, dementia of another. And yet it is certainly instructive to be reminded that the conditions under which these various symptom-groups occur are of very varied sorts.

Tonnini's experiments bear out the observations of Goltz that removal of the occipital lobes is to a considerable extent equivalent in its results to removal of the frontal lobes. In all these cases, the individual strives with force to reëstablish an equilibrium which will answer the same purpose with the old one even though less efficiently. Only there comes a time when this is no longer possible, and this moment arrives especially soon in the case of toxic or nutritive disorders, by which the functions of the whole brain are more or less impaired, even though but slightly,[7] instead of there being simply a reduction in the number of available elements and connections.

One observation of Goltz, cited by Loeb, is especially interesting in connection with the subject of commissural disease. He found, namely, that removal of the anterior portions of both hemispheres caused more dementia than that of a much larger part of one hemisphere, even to the whole hemisphere. Loeb suggests, as a possible explanation, that since the mechanisms of the two hemispheres more or less duplicate each other, one set might be spared without causing so great a disruption of the sense of personality as would follow the loss of the same parts of both sets, and thus a removal of structures that cannot be duplicated.

It should be said here that in spite of the very high value of Loeb's physiological work his psychological deductions with regard to "associative memory," which he makes the basis of consciousness, are by no means free from flaw.

Finally, the recent anatomical studies of v. Monakow should be called to mind, especially as a counterpart to those of Mellus, from which it appears that Flechsig's views are not well grounded when he draws a sharp distinction between the projection-tract areas of the cortex and the association centers, based on the time of formation of the myelin. The situation is far more complex than this, and it is probable that well-defined differences in function between different cortical areas, in Flechsig's sense, do not exist.

If this doctrine is correct, which recognises in the brain only a series of sensori-motor mechanisms, a multitude of threads stretched from periphery to periphery, and as devoid of the power of the storage of memories; and if it is true that there is no exclusively "intellectual" center, then the problem as to how it happens that tumors of the corpus callosum are so prone to cause symptoms of mental defect becomes easier of solution. We do not need to enquire why such lesions do not cause disorders of motion, disorders of cerebral reflexes, because this is just what they do effect, though these losses are in a measure made good by compensation. The sole function of the brain consists either in bringing about actual motion or nascent motions. It exists for nothing else; and mental life consists in clothing these actions, which seem to be concerned only with the needs of the present and the future, with the colors and forms drawn from the life of the past. Into each act of the present the whole of the actor's past enters in some measure.

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[7] Compare Bergson.

Mental disease means the failure on the part of these cerebral reflexes to recur in such a form that the memories of the past can insert themselves in the manner consistent with what had been their previous wont.

But if the "storage of memories" is not a function of the cortex, then it is not a function of the nerve-cell, and in that case the doctrine becomes more reasonable that sees in the neurone an organ of transmission alone.

The following tables contain the records of thirty-eight cases of tumor implicating the corpus callosum.

SYMPTOMS	KNAPP NO. I	KNAPP NO. II	ZINGERLE	SINKLER	BLACKWOOD	PUTNAM NO. I
Anterior portion	Present			Present		Present
Middle portion	Present	Present	Present	Present	Present	Present
Posterior portion	Present	Present		Present	Present	Present
Origin apparently in Corp. Callos.	Yes	Yes	Yes		Yes	Yes
Origin outside but involving Corp. Callos.				On the left side in the Centrum Semi-Ovale		
Tumor confined almost to Corp. Callos.	Yes					
Neighboring parts of brain involved	Flattening of convolutions in both hemispheres. Corpus Striatum and Basal Ganglia flattened. A hemorrhage was present posterior to the tumor in the right hemisphere	Flattening of the convolutions of both hemispheres. Posterior half and the pillars of the Fornix involved. Also the right Gyrus Hippocampus. Extension into the right hemisphere one centimeter, into the left one one-half centimeter	Extension into Septum Pellucidum, into both side Ventricles and especially on the right side the tumor invades the right forceps and extends back into the third ventricle. Both Corp. Quad. flattened, especially the right	Extends into the anterior horn and roof of the left Lateral Ventricle; also right Frontal lobe and extreme tip of right Caudate Nucleus, and above this it includes the roof of the right Lateral Ventricle	A mass the size of a pigeon's egg on either side above the Lateral Ventricles and partly including them. Basal Ganglia not involved	Extends into the right Frontal lobe. Both Lateral Ventricles were almost filled and both Hemispheres invaded. The Basal Ganglia were compressed. The Internal Capsule is compressed but not invaded
Size of tumor		Medium	Medium	Large	Medium	Large
Microscopical Diagnosis	Large	Sarcoma	Sarcoma	Carcinoma (?)	Glioma	Glioma
Remarks		Both Lateral Ventricles dilated. Basal Ganglia not compressed	A few dorsal fibers of the Corp. Callos. remained intact	Small filbert-size tumor mass also found in the posterior portion of the left Centrum Semi-Ovale		

SYMPTOMS	GLÄSER NO. I	GLÄSER NO. II	GLÄSER NO. III	KÖSTER NO. I	KÖSTER NO. II	BRUNS NO. I
Age and sex	Male 72	Woman, 30	Woman, 60	Male, 59	Male, 68	Male, 77
Mental condition in early stages	Intellect clear No complaints made	Memory weak	Intelligence much disturbed Insomnia	Intelligence apparently normal	Loss of memory. Hallucinations of sight and taste	Decreased intelligence. Loss of memory. Irritable
Mental condition in late stages	Remained the same. No impairment of intellect	Intellect clear	Hallucinations Delirium Coma	Increased mental dulness, then apathy and coma	Great disturbance of the intelligence, then stupidity and coma	On admission to hospital insane and wild. Later lassitude and apathy. Coma
Speech	Not noted	Slightly affected and difficult	Not noted	Irrational at first	Not noted	Not noted
Physical inertia	Not noted	Not noted	Not noted	Not noted	Not noted	Not noted
Convulsions	Not noted	First general and then left side only. Head turns to right	Not noted	Not noted	Not noted	No epileptic attacks
Incoordination	None	Not noted	Not noted	When standing tends to fall backwards	Walking and standing became more difficult	Walking became difficult. Often dragged the right foot.
Paresis or Paralysis	Weakness of the left leg greater than the right. No paresis	Paralysis of left extremities and face muscles	At first weakness in right leg, then paralysis in right arm, face and leg.	Rigidity of leg muscles only. No evident paresis of extremities	Cannot raise legs while lying in bed. No evident paresis	Contracture and and paralysis of the right arm and leg. No paralysis of face

Cutaneous Sensibility	Normal	Not noted	Not noted	Somewhat decreased on body and arms, but can localize well	Not noted	Abolished on right side of body
Knee-jerk	Stiffness in both knees. Right knee-jerk normal	Not noted	Not noted	Decreased	Absent	Normal
Optic Neuritis	Not noted	Not noted	Present (?)	Present	Present	None
Vomiting	Not noted	Not noted	Present	Not noted	Present	None
Dizziness	Not noted	Not noted	Not noted	Not noted	Present	None
Headache	Not noted	Present	Present	Not noted	Present	Present
Appearance of general symptoms early late	Not noted	Not noted	Yes	Not noted	Yes	Yes
Condition of pupils	Not noted	Pupils equal and moderately dilated during convulsions	Not noted	Both pupils much contracted	Pupils react to light	Pupils equal and contracted
Cranial Nerves	Not noted	Conjugate deviation of eyes to the right during convulsions.	Not noted	Strabismus divergens	Not noted	Not noted
Mode of death	Not noted	Very sudden.	In coma	In coma	In coma	In coma
Duration of symptoms	About 1 month	Not noted	Not noted	Not noted	Not noted	About 6 months
Remarks						Increasing bodily weakness. Ivol. evacuations

SYMPTOMS	BRUNS NO. II.	BRUNS NO. III.	PONTOPPIDAN	ERB	ZALESKI	RANSOM
Age and sex	Male, 49	Male, 60		Male, 61	Male, 60	Woman, 24
Mental condition in early stages	Loss of memory. Irritable, insomnia and a feeling of fatigue	Mental apathy. Sat perfectly still many hours at a time	Not noted	Hemorrhage into Corp. Callos. Very slight affection of the intelligence	Mental weakness	Irritable and very excitable. Semi-maniacal
Mental condition in late stages	Mental dulness	Great mental dulness	Not noted	The same	Somnolence and apathy	Intelligence intact. Restlessness subsided under treatment
Speech	Somewhat affected	Would not reply to questions but apparently understood.	Not noted	Unaffected	Not noted	Normal
Physical inertia	Not noted	Present	Not noted	Not noted	None	Not noted
Convulsions	Just before death fell down suddenly unconscious	No epileptic attack	Daily epileptic attacks	No tremor	Not noted	Epileptic attacks quite frequent
Incoordination	Not noted	Ataxia. Takes one step forward and then one back	Not noted	No ataxia	Slight ataxia	At times a slight tendency to stagger to left, otherwise gait was steady
Paresis or Paralysis	Paralysis of both arms and legs came suddenly and late during convulsion	Sl. Paresis of Facialis with the exception of frontal branch. Tongue lay immovable	Monospasmus briachialis. Monocontracture	None	Stiffness of all the extremities and neck muscles. Slight paresis right extremities	Doubtful paresis of left angle of mouth. elsewhere none. Head and eyes turn to left

Cutaneous Sensibility	At first normal. Abolished during convulsion	Much decreased	Not noted	Normal	Normal	Lost on legs only, otherwise normal
Knee-jerk	Normal	Normal	Not noted	Absent (probably due to meningitis). Other reflexes were normal	Increased	Normal. Double ankle clonus
Optic Neuritis	No examination	None	Rise of temperature at end, otherwise no general symptoms	Not noted All these symptoms caused by gen. disease	Not noted	Present
Vomiting	Present (late)	None		Present	Present (late)	Present
Dizziness	Present (early)	Not noted		Present	Present	Not noted
Headache	None	None		Present	Present	Present
Appearance of general symptoms early		Not noted		Yes	Yes	Yes
late	Yes					
Condition of pupils	Left pupil larger than right	Equal at first Good reaction to light. Later L. larger than R.		Pupils dilated and do not react. At death pupils were equal in size	Pupils react well to accommodation but not to light	At first normal. At one time after epileptic attack both pupils dilated and of equal size
Cranial Nerves	Normal	Not noted		Normal	Not noted	Normal
Mode of death	During attack of convulsions	Oedema of lungs		Cerebro-spinal meningitis Sub-ac.	Not noted	Not known, but very sudden
Duration of symptoms	About 3 months	About 5 weeks		About 3 weeks	About 3 weeks	About 2 years

SYMPTOMS	DEVIC & PAVIOT	KNAPP NO. I	KNAPP NO. II	GIESE	GREENLESS	M'GUIRE
Age and sex	Male, 46	Male, 42	Male, 58	Male, 53	Male, 68	Male, 31
Mental condition in early stages	Loss of memory and lessened power of attention. Irritable	Failing memory, delirium and violence	Loss of memory and mental power	Loss of memory	Loss of memory and dulness. Takes no notice of things happening about him	Hebetude and dementia characteristic of syphilis
Mental condition in late stages	Early and severe dementia. Somnolence	Not noted	Delirium with mental confusion, dementia and coma	At first wanted to walk and work. Later had no such desire. Depressed and elevated alternately. Apathy and coma	Progressive dementia. Typhoidal state. Coma	Coma
Speech	Normal	Not noted	Not noted	Speech slow, difficult and uncertain	Incoherent	Not noted
Physical inertia	Not noted	Not noted	Not noted	Not noted	Not noted	Not noted
Convulsions	Epileptic attacks beginning six or seven years before other symptoms	General tremor with temporary helplessness	Epileptiform attacks with vertigo	Epileptic attacks. General weakness with great decreased muscular force in legs	General clonic convulsions. "Quivering fits" affecting arms and legs. Paraplegia	General Convulsions
Incoordination	Not noted	Staggers probably from weakness in knees	Inability to walk	Present	Staggers. Cannot stand alone	None
Paresis or Paralysis	During convulsions eyes turn up and to the left. Left hemiplegia especially in legs. Slight contracture of left arm and some in right leg	General impairment of the muscular power	Impairment of strength of left leg. At first no paresis of face muscles. Later paralysis of left arm, face, leg and tongue	Tongue deviates to right. Tremor of face muscles in speaking, paralysis of arms and legs increased	None	Left facial paralysis. Deviation of tongue to left

Cutaneous Sensibility	Normal	Normal	Numbness and prickling feeling in hands	Normal	Not noted	Normal
Knee-Jerk	Normal	Normal	At first normal. Later increased after hemiplegia	Increased	Not noted	Somewhat increased
Optic Neuritis	Vision is good	None	None	Present (slight)	Not noted	None
Vomiting	Not noted	Present	Not noted	Present	Present (late)	Nausea (late)
Dizziness	None	Not noted	Present (first symptom)	Present	Not noted	Not noted
Headache	Not noted	Present	Present	Present	None	Present
Appearance of general symptoms early	Not noted	Yes	Yes	Yes		Yes
late					Yes	
Condition of pupils	At first pupils were normal, then dilated and finally contracted	Pupils dilated and react to light	Not noted	Right pupil larger than left. Reacted slowly	At first were normal with normal reaction. Later reaction was sluggish	Normal
Cranial Nerves	Normal	Slight impairment of nerves of right eye	Not noted	Normal	Normal	Normal
Mode of death	Exhaustion	Not noted	In coma	In coma	Exhaustion	In convulsion
Duration of symptoms	About 11 years	3 months	1 month	About 9 months	Probably 5 years	1 year
Remarks	Incontinence of urine		Involuntary evacuations of urine and feces		General weakness	

SYMPTOMS	BRISTOWE NO. I	BRISTOWE NO. II	BRISTOWE NO. III	BRISTOWE NO. IV	BERKLEY	FRANCIS & STARR
Age and sex	Male, 46	Male, 41	Male, 51	Woman, 25	Male, 45	Woman, 45
Mental condition in early stages	Intelligence impaired. Stupidity	Mental dulness.	On entrance to hospital drowsy. Drowsiness and stupidity	Occasional optic hallucinations. Otherwise intelligent	Monomania. Very loquacious. Intelligence unimpaired	Sluggish. Apathy
Mental condition in late stages	Drowsiness and coma	Stupidity and apathy	Drowsiness increased	Emotional and joyous, then drowsiness and coma	No coma	Not noted
Speech	Did not speak; probably on account of drowsiness	At first little affected. Later aphasia	At first slow and pertinent. Later aphasia	Drawling and indistinct	Unimpaired	During attacks speaks slowly and very seldom
Physical inertia	Present	Present	Present	Present	Not noted	Not noted
Convulsions	No epileptic attacks	No epileptic attack	None	None	Not noted	Attacks of unconsciousness every few weeks, not epileptic
Incoordination	Cannot walk or even stand alone	None	None	On standing tends to fall backwards	None	None
Paresis or Paralysis	Progressive paralysis of right arm and leg. Doubtful paresis of one or both sides of face. No paresis of tongue	Complete hemiplegia of right side involvement of tongue and face muscles	Progressive paral. of left arm and leg. Some rigidity. Doubtful paral. left side of face.	Paral. of left arm and leg with rigidity. Weakness of left side of face and tongue.	None	Occasional twitching of left face. Great tendency to turn head to right (late)
Cutaneous Sensibility	Not noted	Normal. Later a slight hemianesthesia	Not noted	Not noted	Normal	Not noted

Knee-jerk	Not noted	Normal and at last right knee-jerk was increased	Not noted	Not noted	Normal	Normal
Optic Neuritis	Defective vision (?)	Present	None	Failing vision	Vision normal	Present (late)
Vomiting	Nausea	None	None	Present	None	Present
Dizziness	Present	Not noted	Not noted	Present	Not noted	None
Headache	Present	Present	Not noted	Present	None	Present
Appearance of general symptoms early late	Not noted	Not noted	Not noted	Yes	Not noted	Yes
Condition of pupils	Pupils normal. Left eyelid did not close as perfectly as right one.	At first contracted and equal. At death left pupil larger than right	Normal	Pupils equal but no reaction to light and accommodation	Not noted	Right pupil dilated, no reaction in either pupil
Cranial Nerves	Normal	Hearing imperfect on the right side.	Normal	Hearing and taste normal. Decreased sense of smell (?)	Normal	Slight strabismus convergens of right eye
Mode of death	Exhaustion	Exhaustion	Not noted	Not noted	Pulmonary edema	Exhaustion
Duration of symptoms	14 weeks	10 weeks	About 12 weeks	16 months	22 days. Probably lasted several years	9 1-2 months
Remarks	Involuntary evacuations		Involuntary evacuations	Involuntary evacuations		Great general weakness

In all 4 cases there was a tendency to have food remain indefinitely in mouth.

SYMPTOMS	LUTZENBERGER	SEGLAS& LONDE	OLIVER	LABBÉ	PASTURAUD	SCHUPFER
Age and sex	Male, 34	Not noted	Woman, 43	Male, 37	Woman, 56	Woman, 74
Mental condition in early stages	Loss of memory. Irritability	Hallucinations of sight and hearing. Self accusation. Delirium	Hallucinations very emotional. Sensory Illusions	Loss of memory. Sad and apathetic	Whimsical or slightly foolish. Mental dulness	Acute mental disturbance. Psycho-motor agitation
Mental condition in late stages	Coma	Typhoidal and later meningeal state	Sluggish. Hebetude and coma	Light stupor	Apathy. Increased after entrance into hospital	Not noted
Speech	Affected	Not noted	Normal	Speaks slowly and very seldom	Normal	Not noted
Physical inertia	Not noted	Not noted	Not noted	Not noted	Not noted	Not noted
Convulsions	Not noted	Not noted	Not noted	Epileptic attacks	Not noted	Clonic convulsions of right arm and leg.
Incoordination	On standing tends to fall backward	Not noted	None	None	Not noted	Not noted
Paresis or Paralysis	None, even after apoplectic attack	Weakness and stiffness at first of left arm and leg, then later right arm and leg	None	None	Head turned to the right	Paresis and contracture of right arm and leg. Facial muscles normal
Cutaneous Sensibility	Not noted	Not noted	Normal	Normal	Hyperesthesia	General analgesia (?)
Knee-jerk	Absent	Increased	Not noted	Normal	Not noted	Increased and later diminished

Optic Neuritis	Failing Vision		Neuro - Retinitis both eyes, worse in left. Vision obliterated in left eye.	No examination	No examination	No examination
Vomiting	No general symptoms	No general sympcoms	None	Not noted	Present	None
Dizziness			None	Present	Not noted	Not noted
Headache			Not noted	Present	Not noted	None
Appearance of general symptoms early late			Yes	Early	Not noted	Not noted
Condition of pupils			Left pupil larger than right. No reaction in left. Sluggish in right	Not noted	Normal	Normal
Cranial Nerves	Hearing somewhat affected		Smell much affected. Taste and hearing somewhat affected	Not noted	Normal	Normal
Mode of death	Exhaustion		Exhaustion	Exhaustion	Exhaustion	Progressive exhaustion
Duration of symptoms	1 1-2 months	3 weeks	Abou 3 1-2 years	Not noted	16 days	3 to 4 months
Remarks	Apoplectic attack quite early		The symptoms indicated a disease of the base of brain			

SYMPTOMS	D'ALLOCO	PICK NO. I	PICK NO. II	ZINGERLE	SINKLER	BLACKWOOD
Age and sex	Male, 63	Male, 27	Male, 77	Male, 50	Male, 50	Male, 56
Mental condition in early stages	General and mental weakness	Dulness and apathy	Progressive mental weakness	Slight alteration of character. Drowsiness, then rapid onset of mental and physical failure	Silent and morose. Loss of sexual desire. Neurasthenia and hyperchondriacal.	Diminished intelligence. Poor memory Vague fears
Mental condition in late stages	Not noted	Not noted	Not noted	Confusion. Finally stupor and coma. 10 days' duration	Dementia. Indecent and indifferent. Apathy, Coma, with stertorous breathing	Drowsiness with gradually increasing coma
Speech	Disarthria Amnesis aphasia	Not noted	Disturbed	At first unaffected. Later impairment of articulation	Unaffected	Not noted
Physical inertia	Not noted	Not noted	Not noted	Present	Present	Not noted
Convulsions	Not noted	General convulsions	General convulsions	None	None	None
Incoordination	Not noted	Present	Not noted	Disturbance of equilibrium	Not noted	"Lurching gait"
Paresis or Paralysis	Progressive paralysis of right arm and leg. Paresis of left arm, leg and tongue	Paralysis and contracture of right facial muscles. Head turned to right. Paresis of left face with deviation of tongue. Weakness of arms and legs	Paresis of left arm	Widespread but slight paresis of both sides, especially the right side	None	Tremulousness of lips and limbs. Great feebleness. No paralysis

Cutaneous Sensibility	Hemianesthesia of right side	Normal	Normal	Absent	Normal	Not noted
Knee-jerk	Absent	Increased	Not noted Clonus right ankle	Increased especially left. Hypertonicity of muscles of extremities	Increased	Not noted
Optic Neuritis	Present	Present	None	Present	Present	Apparently absent (Not noted)
Vomiting	Present (late)	Present	None	Not noted	None	
Dizziness	Not noted	Not noted	Not noted	Not noted	None	
Headache	Present	Present	None	Not noted	None	
Appearance of general symptoms early	Yes	Yes	Not noted			
late				Yes	Yes	
Condition of pupils	Not noted	Not noted	Not noted	Pupils equal and reacted slowly to light	Pupils contracted and acted sluggishly. G. P. (?)	
Cranial Nerves	Slight Ptosis	Diplopia intermittans	Normal	Normal	Normal	Normal
Mode of death	General progression of symptoms	Not noted	Pneumonia	In coma	In coma	In coma
Duration of symptoms	6 weeks	Not noted	17 months	9 weeks	3 months	5 weeks
Remarks						

SYMPTOMS	PUTNAM NO. I	PUTNAM NO. II	WILLIAMS
Age and sex	Male, 50	Male, 56	Woman, 22
Mental condition in early stages	Lack of mental balance suggesting paresis	Peculiar behavior followed soon by delirium with intervals of clearness	Intellect clear
Mental condition in late stages	Impairment of verbal expression. Failure of memory. Apathy and mental dulness	Coma	Mental apathy and coma
Speech	Awkward and imperfect	Unaffected	Unaffected
Physical inertia	Present	Present (late)	None
Convulsions	Two years before other symptoms, as epileptiform convulsions. None later	None	None
Incoordination	Disturbed equilibrium. Ataxia indicated but doubtful (Not typical)	Gait uncertain and unsteady	Slight staggering (early symp)
Paresis or Paralysis	Paresis of right hand and right side of face with disturbance of speech. Later weakness of legs	Paresis of left leg right leg (not right arm)	None
Cutaneous Sensibility	Temporary paresthesia of right leg coming suddenly and very early	Not noted	Not affected at first. Value of later examination impaired by mental condition
Knee-jerk	At first normal. Later both exaggerated	Diminished and finally abolished	Right knee-jerk absent. Left knee-jerk normal
Optic Neuritis	Present (late)	Not noted	Present
Vomiting	Present	None	Present
Dizziness	Present	None	Present
Headache	Present	None	Present
Appearance of general symptoms early		Yes	Yes
late	Yes		
Condition of pupils	Normal	Not noted	Left pupil temporarily dilated. No reaction in either pupil
Cranial Nerves	Normal	Normal	Normal
Mode of death	Gradual failure	In coma	In coma
Duration of symptoms	About 1 year	3 weeks	15 months
Remarks			

SYMPTOMS	BRISTOWE NO. I	BRISTOWE NO. II	BRISTOWE NO. III	BRISTOWE NO. IV	GLÄSER NO. I	GLÄSER NO. II
Anterior portion	Present	Present	Present	Present	Present	Present Whole of right side destroyed
Middle portion			Present	Present		Present
Posterior portion						Present
Origin apparently in Corp. Callos.	Yes	Yes	Yes		Yes	
Origin outside but involving Corp. Callos.				Corpus Striatum and Thalamus Opticus		Right Hemisphere and pressing especially on anterior portion of Corp. Callos.
Tumor confined almost to Corp. Callos.	Yes				Yes	
Neighboring parts of brain involved	Slight involvment of Centrum Ovale chiefly on left side	Centrum Ovale chiefly left side. Left 2d and 3d frontal convolutions. Left ascending parietal convolutions	Anterior two-thirds of Fornix extending into right hemisphere	Distension of right ventricle and displacement of Fornix anl Septum Lucidum	Very slight invasion into both hemispheres	Chief part of tumor in the covering of 3d and right lateral ventricles and also in right hemisphere
Size of tumor	Small	Small	Small	Large	Medium	Large
Microscopical Diagnosis	No exact microscopical diagnosis made. Probably all Sarcomata.					
Remarks	In first three cases tumor arose obviously in Corp. Collos. and extended symmetrically along radiating fibers of Corp. Callos. into white matter of Hemispheres					Left posterior Cornu much dilated. On left side tumor ends also in covering to lateral ventricle

	GLÄSER NO. III	GIESE	BERKLEY	BRUNS NO. I	BRUNS NO. II	BRUNS NO. III
Anterior portion			Present			Present
Middle portion				Present	Present	
Posterior portion	Present	Present		Present	Present	
Origin apparently in Corp. Callos.		Yes	Yes	Yes	Yes	
Origin outside but involving Corp. Callos.						In first Frontal convolution
Tumor confined almost to Corp. Callos.			Yes	Yes		
Neighboring parts of brain involved		Slight invasion of right Occipital lobe. Posterior portion of tumor joined to Falx and Tentorium. Left Corpus Quad. slightly flattened and softened	A few apparently sclerotic fibers passed to Centrum Ovale	Invasion of right side into Precuneus. On left side into white substance on Hemisphere over Lateral Ventricle	On both sides. Behind it includes Forceps posterior and goes into posterior horns of both Lateral Ventricles, also Precuneus on both sides	Pressure on first Frontal convolution and Gyrus Fornicatus. Second tumor in knee of Corp. Callos. Third tumor, small, over Nucleus Caudatus
Size of tumor	Medium	Small	Small	Medium	Large	Small
Microscopical Diagnosis		Glio-Sarcoma				
Remarks		Edge of Gyrus Fornicatus free on both sides	Tumor situated in middle line in Corp. Callos.		Fornix and Cornu Ammonis remained free	There were three very small tumors situated very close together in region of anterior portion of Corp. Callos.

SYMPTOMS	ZALESKI	KÖSTER NO. I	KÖSTER NO. II	FRANCIS & STARR	LUTZENBERGER	OLIVER
Anterior portion		Present		Present		Present
Middle portion		Present	Present		Present	
Posterior portion	Present	Present	Present			
Origin apparently in Corp. Callos.	Yes	Yes	Yes	Yes	Yes	Yes
Origin outside but involving Corp. Callos.						
Tumor confined almost to Corp. Callos.	Yes					Yes
Neighboring parts of brain involved	Slight invasion into white substance of left hemisphere	Invasion to Centrum semiovale on side. Beneath invasion Septum pelucidum, Fornices and upper part of Corp. Callos. remained free	Includes entire Splenium and part of Fornix lying beneath it. On left side invasion into white substance of Hemisphere above Lateral Ventricle. No sharp border	Both Frontal lobes much invaded	Invasion of cortex and left hemisphere. Also into edge of Gyrus Supra - Marginalis	No gross lesion of other parts of brain. Left optic nerve compressed
Size of tumor	Medium	Large	Large	Large	Large	Small
Microscopical Diagnosis	Glioma	Sarcoma	Glioma	Neuro-Glioma	Glioma	Sarcoma
Remarks		Both Ventricles distended with fluid				

SYMPTOMS	D'ALLOCO	PICK NO. I	PICK NO. II	SCHUPFER	PASTURAUD	LABBÉ
Anterior portion	Present	Present			Present	Present
Middle portion	Present	Present	Present			
Posterior portion	Present			Present		
Origin apparently in Corp. Callos.		Yes	Yes	Yes		
Origin outside but involving Corp. Callos. Tumor confined almost to Corp. Callos.	Left hemisphere				In fissure between Frontal lobes	Left and right Frontal lobes connected by a bridge
Neighboring parts of brain involved	Right Frontal lobe, whole of left internal Capsule, left Hemisphere	Cavity in posterior Cornu and the porterior portion of anterior Cornu of the right side	Fornix involved	Cornu Ammonis and Splenium Pulvinar	Left Frontal lobes	All three Frontal lobes more involved than the right ones
Size of tumor	Large	Medium	Small	Small	Small	Large
Microscopical Diagnosis	Not noted	Sarcoma	Glio-Sarcoma	Not noted	Sarcoma	Glioma
Remarks			Hydromyelia			

SYMPTOMS	M'GUIRE	GREENLESS	PONTOPPIDAN	ERB, HEM. C. C.	RANSOM	DEVIC & PAVIOT
Anterior portion	Present	Present	Present (?)	Present Only lowest layers not entirely destroyed. These are recognised filled with blood	Present	Present
Middle portion				Present		
Posterior portion				Present		
Origin apparently in Corp. Callos.	Yes	Yes	Yes	Yes	Yes	Yes
Origin outside but involving Corp. Callos.						
Tumor confined almost to Corp. Callos.			Yes		Yes	
Neighboring parts of brain involved	Had made an excavation in the Fornix. Both Hemispheres involved more on the left side. Slight excavation in Cortex above on the left side	Fully one-third of the tumor was anterior to the Corp. Callos. in left Frontal lobe involving the white matter	Left Hemisphere involved	The lateral, 3d and 4th Ventricles all filled with blood. Cortex engorged with blood and pulpy, as were also the Great Ganglia, Pons, and Cerebellum.	Tumor bulged very slightly into each ventricle, otherwise brain healthy	Invasion of the right Hemisphere up to its superior border, also the Centrum Ovale, Right Frontal and Paracentral lobes. Tumor does not extend outside of Corp. Callos. on the left side
Size of tumor	Small	Small	Small	Large	Small	Large
Microscopical Diagnosis		Glio-Sarcoma	Glio-Sarcoma	Hemorrhage into Corp. Callos.	Sarcoma	Glioma
Remarks	Tumor was very small 3.5x2 centimeters	Cerebrum, otherwise quite normal		Neighboring parts of brain are hardly involved by hemorrhages		

SYMPTOMS	PUTNAM NO. II	WILLIAMS
Anterior portion	Present	Present
Middle portion	Present	Present
Posterior portion	Present	Present
Origin apparently in Corp. Callos.		Yes
Origin outside but involving Corp. Callos.	In the right hemisphere	Possible origin in the region of the Lamina Terminalis
Tumor confined almost to Corp. Callos.		
Neighboring parts of brain involved	The right Hemisphere involved to within 1 1-2 centimeters of its upper surface. Left Hemisphere not invaded. The tumor has a maximal size at level anterior to optic chiasm. The Fornix is involved in part as it lies opposed to Corp. Callos.	Both Lateral Ventricles almost obliterated. A small mass extends down into the Interpeduncular space and compresses the second and third nerves
Size of tumor	Medium	Large
Microscopical Diagnosis		Glioma
Remarks	Neither the Lateral nor third Ventricles are involved.	Some increase in intraventricular fluid.

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